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Long-Term Follow-Up of Right Ventricular Monomorphic Extrasystoles

Fiorenzo Gaita, MD,* Carla Giustetto, MD,† Paolo Di Donna, MD,* Elena Richiardi, MD,* Luigi Libero, MD,† Maria C. Rosa Brusin, MD,† Giuseppe Molinari, MD,* Giampaolo Trevi, MD†

Asti and Torino, Italy

OBJECTIVES The purpose of this study was to verify in a long-term follow-up whether frequent monomorphic right ventricle extrasystoles may progress to arrhythmogenic right ventricular dysplasia (ARVD).

BACKGROUND Frequent monomorphic right ventricle extrasystoles are generally considered benign. However, in patients with this pattern, cardiac magnetic resonance (MR) has recently shown anatomical and functional abnormalities of the right ventricle.

METHODS Sixty-one patients who had been classified by noninvasive examinations as having frequent idiopathic right ventricle ectopy were contacted after 15 ± 2 years (12 to 20) and submitted to clinical examination, electrocardiogram (ECG), Holter monitoring, stress test, signal averaged ECG, echocardiography and, in 11 patients, cardiac MR. The primary end point was to ascertain the presence of cases of sudden death or progression to ARVD.

RESULTS At the end of the follow-up, 55 patients were alive; six died, none of sudden death; eight stated to be well but refused further examinations. The 47 patients examined had normal ECG; in 24 patients (51%), extrasystoles were no longer present at Holter monitoring; late potentials were present in up to 15% of the patients; the right ventricle was normal at echocardiography. In 8 of 11 patients (73%), cardiac MR showed focal fatty replacement and other abnormalities of the right ventricle.

CONCLUSIONS In this long-term follow-up study, no patient died of sudden death nor developed ARVD; two-thirds of the patients were asymptomatic, and, in half of the patients, ectopy had disappeared. Focal fatty replacement in the right ventricle was present in most. (J Am Coll Cardiol 2001;38:364–70) © 2001 by the American College of Cardiology

Ventricular extrasystoles are a common finding in patients with and without heart disease. In the presence of heart disease, frequent and repetitive extrasystoles are an independent predictive factor of total mortality and sudden death (1–3). On the other hand, very frequent monomorphic ventricular complexes and even bursts of ventricular tachycardia in subjects without evidence of heart disease are generally considered benign. In 1922, Gallavardin (4) first described this kind of arrhythmia. In 1969, Rosenbaum (5) defined ventricular ectopy with left bundle branch block (LBBB) morphology and the main QRS forces directed inferiorly as "typical for normal subjects." This kind of extrasystole is more frequent during the day than it is at night and is transiently suppressed by sinus tachycardia (6). The site of origin is most often the right ventricular outflow tract and, to a lesser extent, the interventricular septum (7). In the majority of these patients, echocardiography does not identify structural cardiac abnormalities (8). More recently, however, using cardiac magnetic resonance (MR) imaging, a high prevalence of fatty replacement and other anatomic and functional abnormalities has been demonstrated in patients with apparently idiopathic right outflow tract tachyarrhythmias (9–11). A partial overlap exists between the area involved in arrhythmogenic right ventricular dysplasia (ARVD) and idiopathic right ventricular ectopy. The infundibulum is one of the sites most frequently involved in ARVD, together with the diaphragmatic and apical walls (12). As the latter is a progressive disease (13–16), the question arises whether idiopathic right ventricular ectopy could be an early manifestation of ARVD. Arrhythmogenic dysplasia is easy to recognize when the right ventricle is extensively involved, but initial forms could be missed. Some patients with apparent idiopathic right ventricular arrhythmias, therefore, might be affected at an early stage.

In this study, patients with a diagnosis of idiopathic right ventricular ectopy, established through clinical examination and noninvasive tests, were evaluated after a follow-up of at least 12 years to verify the occurrence of sudden death and the possible evolution towards ARVD.

METHODS

From all patients with ventricular ectopy who were evaluated at the University of Torino between January 1980 and October 1987, we selected those having extrasystoles with LBBB morphology or positive QRS from V_1 to V_6 at 12-lead electrocardiogram (ECG), suggesting a right ventricular or septal origin, in whom no clinical evidence of heart disease was found after routine cardiac examination, including 12-lead ECG, chest X-ray and echocardiography. Patients were referred to our center because of palpitations or the detection of ventricular extrasystoles on a routine ECG. No patient had syncope nor near-syncope. These patients had >40/h (>1,000/24 h) monomorphic ventricular extra-

From the *Department of Cardiology of the Civil Hospital of Asti, Asti, Italy; and the †University of Torino, Torino, Italy.

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Abbreviations and Acronyms

ARVD = arrhythmogenic right ventricular dysplasia
ECG = electrocardiogram
LBBB = left bundle branch block
MI = myocardial infarction
MR = magnetic resonance
RBBB = right bundle branch block
SAECG = signal averaged electrocardiogram

systoles, occurring at sinus rates between a critical upper and lower range, determined by observing the relation between the number of extrasystoles and heart rate variations on Holter monitoring. There were occasional ventricular couplets and triplets in some patients; no more complex ventricular arrhythmias were documented. Ectopy disappeared during stress test. Based on these criteria, a diagnosis of idiopathic right ventricular ectopy was made. All the evaluations had been performed in the absence of antiarrhythmic therapy, including beta-blockers.

These patients were contacted again between May 1999 and February 2000. We obtained a complete medical history, clinical examination, ECG, 24-h Holter monitoring and treadmill exercise test (Bruce protocol). Extrasystoles were considered no longer present if they were <100/24 h. Echocardiography was performed using an Ultramark 9 (Advanced Technology Laboratory, Bothell, Washington) and a Sonos 2000 (Agilent Technologies, Palo Alto, California). Short- and long-axis views were obtained in the left parasternal, apical and subcostal locations. The diameter of the outflow tract of the right ventricle was evaluated in the parasternal short-axis view. Longitudinal and transverse diameter and the area of the right ventricle were taken in the long-axis four-chamber view. Signal averaged ECG (SAECG) was performed using the 1200 EPX Model (Arrhythmia Research Technology Inc., Austin, Texas). Two hundred to 300 beats were averaged to obtain a noise level <0.2 μV. Two high-pass filter settings were used at 25 Hz and 40 Hz (17). Late potentials were considered to be present if two of the three criteria were met. A random subgroup of 11 patients was also investigated with cardiac MR. The study was performed using a 1.5 T superconducting magnet with 25 mT/m gradients (Magnetom Vision, Siemens, Erlangen, Germany). The morphologic study was performed in the short- and long-axis planes through breath hold single slice turbo-spin echo sequences (time of echo 32 ms), with and without spectral fat-suppression. The functional study was performed through velocity flow refocused gradient-echo sequences (cine MR): two long-, two short-axis and one sagittal planes were chosen; a time of echo 4.8 ms and a flip angle of 20° were used (18).

Statistical analysis. Data were expressed as mean ± SD. Student *t* test for paired data was used to compare the number of extrasystoles at enrollment and at follow-up 24-h Holter monitoring. Student *t* test for unpaired data was used to compare age at enrollment between the patients who survived and the group who died. A two-tailed p value of ≤0.05 was considered significant. Survival analysis was performed with the Kaplan-Meier method.

RESULTS

Sixty-one patients (30 women and 31 men) were included in this study. At enrollment, mean age was 44 ± 15 years; 55 complained of palpitations, and 6 were asymptomatic. Twelve-lead ECG was normal, including QT interval, in all patients. The morphology of the extrasystoles was LBBB and vertical axis in 52 patients (85%), LBBB and left-axis deviation in 1 and positive QRS from V_1 to V_6 (right bundle branch block [RBBB]) and left-axis deviation in 8 (Fig. 1). The mean number of extrasystoles/h at 24-h Holter monitoring was 526 ± 456 (range 66 to 2,260). Fifty patients (82%) had ectopy at stress test basal ECG, which disappeared in all with variable degrees of heart rate increase; extrasystoles appeared during recovery in 2 of the 11 patients who did not show ectopy before exercise. Echocardiogram was normal in all but two patients: one patient had mitral valve prolapse without regurgitation, and the other had rheumatic mitral stenosis without hemodynamic relevance. Because of the apparent absence of relationship with ectopy originating from the right outflow tract, these latter two patients were included in the study.

At a follow-up ranging from 12 to 20 years, mean 15 years ± 2 years, 55 patients (90%) were alive, and 6 patients died (10%). Mean age at the first visit was 40 years ± 14 years in the patients who survived and 65 years ± 10 years in the group who died ($p < 0.001$). Survival analysis showed a mortality rate of 3% at 5 years, 7% at 10 years and 10% at 15 years. No patient died more than 13 years after the first evaluation. Of the six patients who died, none died suddenly; one died from lung cancer, one from myocardial infarction (MI), one from stroke, two from multiorgan failure and one in a car accident. Eight patients stated that they were asymptomatic and in good health but declined the invitation to undergo further examinations. None of them was taking antiarrhythmic therapy. Forty-seven patients agreed to be evaluated. The results of the examinations of this group of patients at enrollment into the study and at follow-up are reported in Table 1. Six patients had developed hypertension, and two had developed ischemic heart disease (inferior MI and unstable angina, both treated with revascularization); one patient developed a moderate aortic incompetence. The functional class was unchanged in the patient with the mild mitral stenosis. Only 15 of the 47 patients (32%) still had palpitations. None had been admitted to a hospital or had been otherwise found to have sustained ventricular tachycardia. No patient was taking antiarrhythmic drugs. Twelve-lead ECG was normal in all, except in the one who had an MI during the follow-up. This patient had Q-waves in the inferior leads. At 24-h Holter

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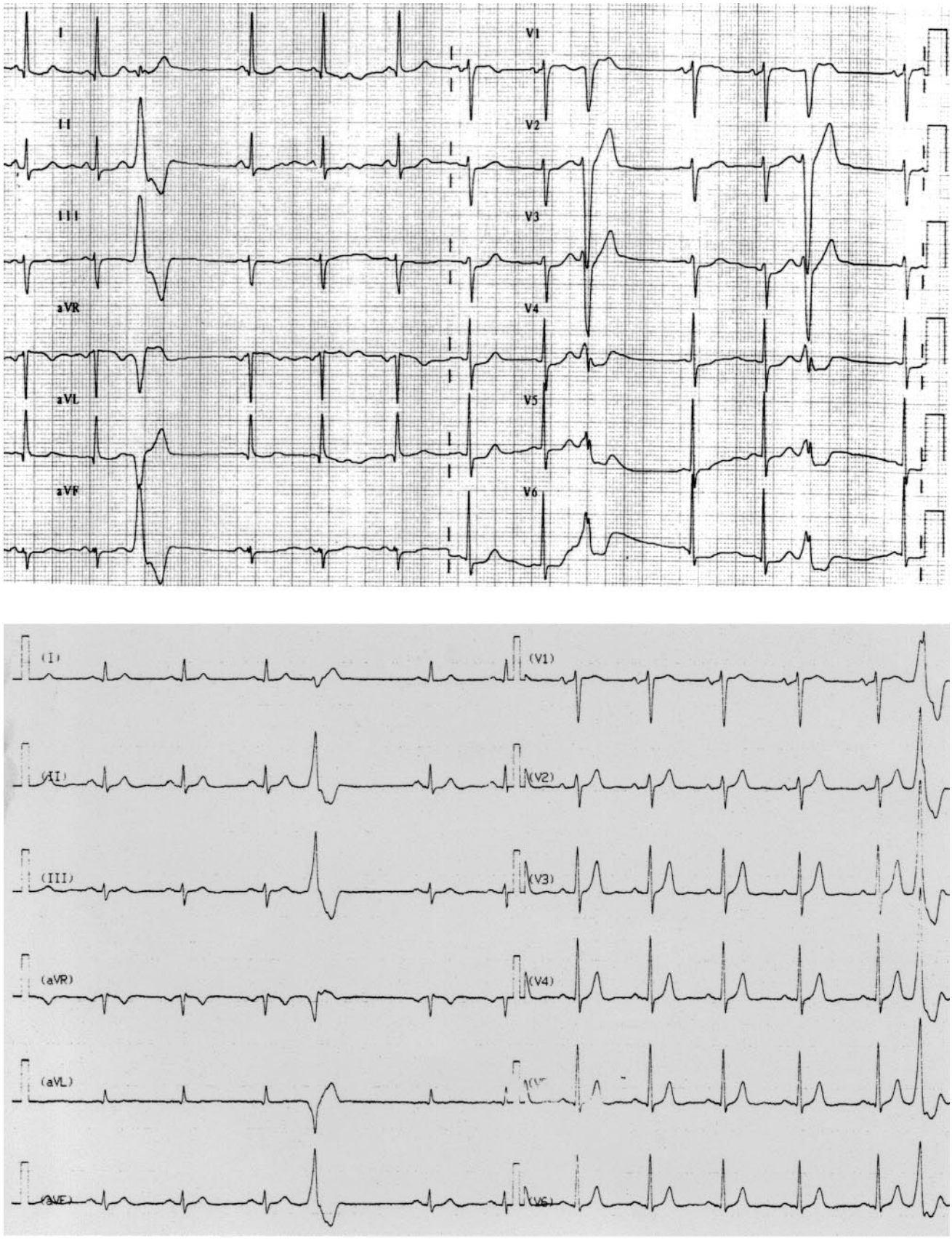

Figure 1. Twelve-lead electrocardiograms showing ventricular premature beats with left bundle branch morphology and vertical axis **(top)** and with positive QRS from V_1 to V_6 **(bottom)**.

Table 1. Patients' Data

	Patients' Data at Enrollment									Patients' Data at Follow-Up							
Number	Pt.	Age	Gender	Symptoms	VPC Morphology	VPC h	VPC 24 h	Stress Test	Echo	F.U.	Symptoms	History	VPC h	VPC 24 h	Stress Test	Echo	SAECG
1	A.C.	29	M	Yes	LBBB, Va	112	2,679	D	N	16	No	N	23	542	A	N	−
2	A.L.	26	M	No	LBBB, Va	170	4,073	D	N	16	No	N	127	3,049	D	N	−
3	B.C.	59	F	Yes	LBBB, Va	66	1,573	R	N	20	No	PAF, Hypertension	0	4	A	AI+++	−
4	B.E.	51	F	Yes	LBBB, Va	193	4,623	D	N	16	Yes	N	2	45	A	N	−
5	B.F.	16	M	No	LBBB, Va	109	2,620	R	N	14	No	N	0	2	A	N	−
6	B.G.	44	M	Yes	LBBB, Va	303	7,283	D	N	20	No	Hypertension	8	190	D	N	−
7	B.L.	42	M	Yes	LBBB, Va	1,136	27,256	A	N	15	Yes	N	523	12,554	D	N	+*
8	B.M.	57	F	Yes	LBBB, Va	211	5,053	D	N	13	No	N	0	2	A	N	−
9	B.M.	36	F	Yes	LBBB, Va	1,485	35,639	D	N	16	Yes	N	283	6,780	P	N	+
10	B.M.	39	M	Yes	LBBB, Va	246	5,909	D	N	15	Yes	N	8	200	A	N	−
11	B.W.	55	M	Yes	LBBB, Va	457	10,956	D	N	15	Yes	CLD	40	956	A	N	−
12	C.A.	29	F	Yes	LBBB, Va	470	11,276	D	N	17	Yes	N	4	100	D	MP	−
13	C.A.	36	M	Yes	LBBB, Va	443	10,642	D	N	12	No	N	0	3	A	N	−
14	C.F.	11	M	No	LBBB, Va	467	11,206	A	N	14	No	N	0	0	A	N	−
15	C.R.	33	F	Yes	LBBB, Va	688	16,518	D	N	13	Yes	N	0	1	A	N	+
16	D.A.	18	M	No	LBBB, La	430	10,328	D	N	15	No	N	236	5,668	R	N	−
17	D.G.	44	M	Yes	RBBB, La	2,260	54,231	A	N	15	No	N	24	569	VT	N	−
18	D.L.	41	M	Yes	LBBB, Va	291	6,990	A	N	15	No	Hypertension	4	95	A	N	−
19	F.G.	47	M	Yes	LBBB, Va	238	5,707	D	N	14	No	Hypertension	25	590	A	N	−
20	F.G.	50	F	Yes	LBBB, Va	1,027	24,652	D	N	13	No	N	5	120	A	N	−
21	F.L.	57	F	Yes	LBBB, Va	930	22,331	D	N	14	No	N	0	0	A	N	−
22	G.F.	49	M	No	RBBB, La	436	10,462	D	N	18	No	N	51	1,233		N	−
23	G.F.	55	M	Yes	LBBB, Va	271	6,500	D	N	16	No	N	0	2	A	N	−
24	G.I.	20	F	Yes	LBBB, Va	235	5,646	D	N	14	No	N	0	3	A	N	−
25	I.C.	31	F	Yes	LBBB, Va	792	19,000	D	MS	12	Yes	N	656	15,755	D	MS	−
26	L.A.	54	F	Yes	LBBB, Va	606	14,541	D	N	15	No	CHD	0	0	A	N	−
27	L.L.	55	M	Yes	LBBB, Va	488	11,700	D	N	16	No	CHD	0	2	D	Inf Akin	−
28	M.A.	36	F	Yes	LBBB, Va	393	9,442	D	N	14	No	N	2	57	R	N	−
29	M.C.	29	F	Yes	RBBB, La	220	5,269	D	N	14	Yes	N	14	345	D	N	+*
30	M.C.	48	M	Yes	LBBB, Va	1,036	24,868	D	N	12	No	N	0	5	A		−
31	M.C.	52	F	Yes	LBBB, Va	1,925	46,201	D	N	15	No	N	0	1	A	N	+
32	M.D.	41	F	Yes	LBBB, Va	472	11,321	D	N	20	Yes	N	6	144	Different	N	−
33	M.E.	46	F	Yes	LBBB, Va	514	12,342	D	N	14	Yes	N	6	135	Different	N	−
34	M.F.	24	M	Yes	LBBB, Va	221	5,312	D	N	15	No	N	0	2	A	N	−
35	M.S.	48	F	Yes	LBBB, Va	506	12,133	D	N	16	No	Hypertension	10	239	P	N	−
36	N.I.	57	F	Yes	LBBB, Va	769	18,456	A	MP	13	Yes	N	868	20,836	P	MP	−
37	N.P.	42	M	Yes	LBBB, Va	856	20,538	A	N	16	No	Hypertension	27	659	P	N	−
38	P.F.	45	M	Yes	RBBB, La	149	3,567	D	N	15	No	N	1	24	D	N	+
39	P.I.	48	F	Yes	RBBB, La	89	2,125	D	N	12	Yes	N	8	200	A	N	−
40	P.L.	37	M	Yes	LBBB, Va	646	15,515	D	N	15	No	N	0	1	A	N	−
41	R.F.	29	F	Yes	LBBB, Va	468	11,233	D	N	13	No	N	0	0	A	N	−
42	T.F.	38	M	Yes	RBBB, La	437	10,491	D	N	13	Yes	N	126	3,014	D	N	−
43	T.R.	30	F	Yes	LBBB, Va	97	2,321	D	N	13	No	N	0	1	A	N	−
44	T.R.	34	F	Yes	LBBB, Va	513	12,323	D	N	12	No	N	0	0	A	N	+*
45	V.R.	8	F	Yes	RBBB, La	125	3,000	D	N	15	Yes	N	98	2,341	D	N	−
46	V.S.	55	M	Yes	LBBB, Va	200	4,800	D	N	14	No	Hypertension	0	5	Different	N	−
47	U.M.	45	F	Yes	LBBB, Va	537	12,890	A	N	13	Yes	N	0	11	A	N	−

A = absence; AI+++ = moderate aortic incompetence; CHD = coronary heart disease; CLD = chronic lung disease; D = disappearance; Different = different morphology; Echo = echocardiogram; F.U. = follow-up; Inf Akin = inferior akinesis; La = left axis; LBBB = left bundle branch block; MP = mitral prolapse; MS = mitral stenosis; N = normal; P = persistence; PAF = paroxysmal atrial fibrillation; Pt. = patient; R = appearance during recovery time; RBBB = right bundle branch block (positive QRS from V_1 to V_6); SAECG = signal averaged electrocardiogram; Va = vertical axis; VPC h = ventricular premature complexes per hour at 24-h Holter monitoring; VPC 24 h = total number of ventricular premature complexes at 24-h Holter monitoring; VT = ventricular tachycardia; + = presence of late potentials; +* = presence only with 40 Hz filter; − = absence of late potentials.

Table 2. Cardiac Magnetic Resonance

Number	Pt	VPCs Morphology	Holter at Follow-Up	Wall Motion	RV Diameters	Fatty Replacement
1	B.M.	LBBB, Va	P	Normal	Normal	Focal, outflow tract and apex
2	B.M.	LBBB, Va	P	Normal	Normal	No
3	C.A.	LBBB, Va	D	Normal	Normal	Focal, free wall
4	C.A.	LBBB, Va	D	Normal	Normal	No
5	C.F.	LBBB, Va	D	Dyskinetic apex	Normal	Focal, apex and free wall
6	D.A.	LBBB, La	P	Normal	Normal	No
7	M.D.	LBBB, Va	P	Normal	Normal	Focal, outflow tract
8	N.I.	LBBB, Va	P	Normal	Normal	Focal, outflow tract and apex
9	P.F.	RBBB, La	D	Normal	Normal	Focal, outflow tract, posterobasal and apex
10	P.I.	RBBB, La	P	Normal	Normal	Focal, right and left ventricle apex
11	U.M.	LBBB, Va	D	Dyskinetic and thin free wall, trabecular disarray	Normal	Focal, outflow tract and posterobasal

D = VPCs disappearance; La = left axis; LBBB = left bundle branch block; P = VPCs persistence; RBBB = right bundle branch block; RV = right ventricle; Va = vertical axis; VPCs = ventricular premature complexes.

monitoring in 24 of the 47 patients (51%), extrasystoles were no longer present (<100/24 h), while 9 patients (15%) still had more than 40 extrasystoles/h; the mean number of extrasystoles/h was 68 ± 176 ($p < 0.001$ vs. enrollment), range 0 to 868.

Stress test was performed in 47 patients: ectopy was absent at resting ECG in 32 (68%); in two of the patients, ectopy appeared at recovery time, and, in three patients, extrasystoles with different morphology were observed during the test. Of the 15 patients (32%) who presented ectopy on the ECG at rest, 10 showed the disappearance of the extrasystoles with variable degrees of heart rate increase. In four patients, the ectopy persisted during the test: the maximal heart rate in these patients was lower than it was in the stress test at enrollment. In one patient who had accelerated idioventricular rhythm with RBBB and left-axis morphology alternating with sinus rhythm at basal ECG, acceleration of the ventricular rate in the range of ventricular tachycardia occurred. After stopping the exercise, the tachycardia slowed down again to idiopathic ventricular rhythm alternating with sinus rhythm.

Signal averaged ECG was obtained in all patients; criteria for late potentials were present in four patients (9%) when a high-pass filter of 25 Hz was used and in seven patients (15%) when the high-pass filter was set at 40 Hz. Echocardiography showed moderate aortic incompetence in a hypertensive patient, inferior akinesia in the patient with MI and a further mitral valve prolapse without incompetence; it was unchanged in the other cases. No abnormalities of the right ventricle were observed.

At cardiac MR (Table 2), only three patients had a completely normal pattern. Focal adipose replacement was found in 8 of 11 patients (three areas in one patient, two areas in five and one area in two) (Fig. 2); right ventricle dyskinesia occurred in two patients; thin right ventricle free wall and trabecular disarray were found in one case each. One patient showed all the described abnormalities: focal fatty tissue replacement of the outflow and posterobasal tract, right ventricle dilation and dyskinetic and thin free wall. This patient had a normal echocardiogram, absence of late potentials and disappearance of extrasystoles at follow-up 24-h Holter monitoring.

DISCUSSION

Ventricular extrasystoles are commonly observed in the ambulatory population. Their prognostic significance depends largely on the presence of heart disease (1–3). Ventricular ectopy originating from the right ventricle in subjects without underlying heart disease has been known for a long time and is generally considered benign. However, cardiac MR imaging has recently shown a high incidence of focal right ventricular abnormalities in this population (9–11). The clinical meaning of these findings is still unclear. Ventricular arrhythmias of right ventricle origin could be the expression of an early form of ARVD. This condition is recognized as a cause of sudden, unexpected death, particularly in younger individuals, and this event may be the first presentation of the disease. In a postmortem study from northern Italy, 20% of 60 patients under the age of 35 years who died suddenly and unexpectedly had histological findings consistent with ARVD (19). Large studies and long follow-up periods are needed to clarify this issue. In 1985, Kennedy et al. (20) published the follow-up study (mean 6.5 years) of 73 apparently healthy subjects with frequent and complex ventricular ectopy. The conclusion was that the long-term prognosis of these patients is similar to that of the healthy population. In our study we report the follow-up lasting from 12 to 20 years for 61 patients with monomorphic right ventricle extrasystoles. No patient developed ARVD; none died suddenly nor had sustained ventricular tachycardia. At the end of the follow-up in two-thirds of the patients, palpitations had disappeared, and none presented ECG alterations suspicious of ARVD (12,21). At 24-h Holter monitoring, the

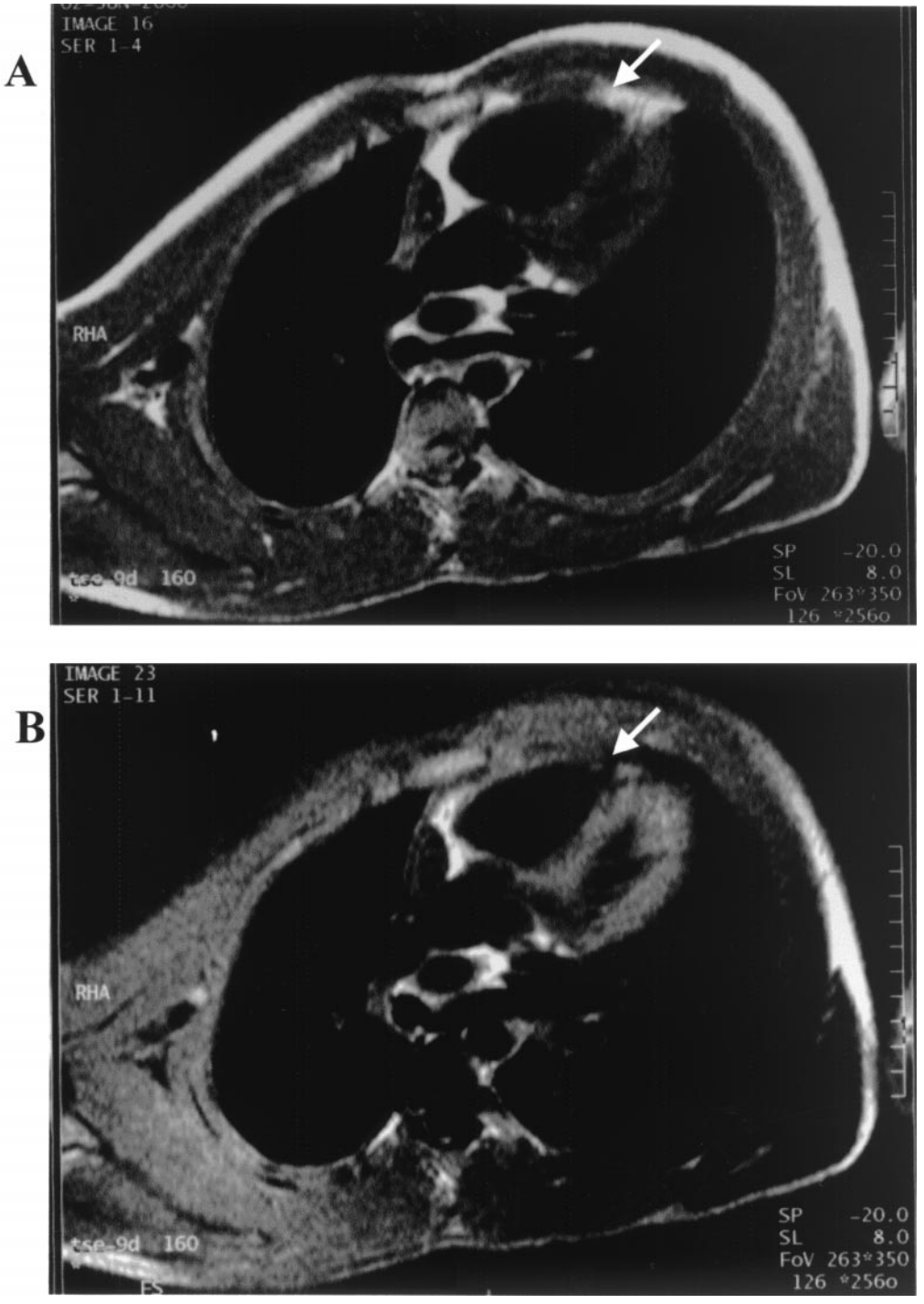

Figure 2. (A,B) Magnetic resonance turbo-spin-echo images obtained at 1.5 T in a patient with monomorphic right ventricular extrasystoles. Long-axis view demonstrating high-signal intensity at the apex of the right ventricle **(arrow)**, without **(A)** and with **(B)** spectral fat saturation. **Image A** acquired without spectral fat saturation, shows high-signal intensity within the right ventricle apical wall due to the presence of fatty tissue. When the same image is acquired with spectral fat saturation **(B)**, the high-signal intensity of fatty tissue disappears, and the fatty replacement is imaged as an area of signal void within the right ventricle.

disappearance of ventricular extrasystoles was observed in half of the population, and stress test confirmed this finding. During echocardiography no abnormalities of the right ventricle were observed. An SAECG showed late potentials in a low percentage of patients. On the contrary, a high sensitivity and specificity of late potentials has been reported in patients with ARVD. The low incidence of ventricular late potentials in our population compared with patients with ARVD may be explained by differences in the arrhythmogenic substrate.

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Cardiac MR imaging. At MR imaging, 8 of the 11 patients showed localized adipose replacements, in contrast with the diffuse pattern that is observed in patients with ARVD (10,16). Cardiac MR has allowed the identification of focal structural changes, which could not have been recognized with echocardiography and have not been observed in normal subjects (10); these abnormalities might be the anatomical substrate of right ventricle extrasystoles. As arrhythmias in these patients had not worsened during a mean follow-up of 15 years, there is a low probability that these focal abnormalities may represent an early manifestation of ARVD, which is a progressive disease (13–16).

Some patients developed a heart disease during the follow-up. However, at enrollment there was no evidence of most of these diseases (hypertension, ischemic heart disease, aortic incompetence), which are probably related to aging. Nonetheless, in the presence of an abnormal substrate, the prognostic value of the ectopy may be different.

When faced with right ventricular premature contractions in an otherwise normal ECG, we have to decide whether it represents a benign pattern or a possible early form of ARVD. In our study after a mean follow-up of 15 years, most of the patients are asymptomatic, showing corresponding reduction and, often, disappearance of the extrasystoles. Moreover, no morphologic change suggestive of progression to ARVD was observed. Based on the data of our study, a normal 12-lead ECG, the monomorphic pattern of ectopy and the heart rate dependence at 24-h Holter monitoring, the suppression with increasing heart rate at stress test and the normal echocardiographic pattern are sufficient to pose the diagnosis of benign right ventricle arrhythmias.

Study limitations. Even if this group of patients is very homogeneous, 61 patients may still not be enough to exclude exceptions to this benign natural history. Moreover, some patients in our population were very young. Even if a mean follow-up of 15 years is a long period of time, it may still be short as compared with the life expectancy of the youngest patients.

The finding of late potentials has been reported in studies on right ventricular outflow tract tachycardia (22). At the present time, the possibility that late potentials may predict the progression to ARVD is not established, and their meaning remains to be defined.

Cardiac MR was performed only in a small percentage of patients because it is not recognized as a routine examination for patients with ventricular extrasystoles without heart disease. The findings are noteworthy for the high prevalence of detected right ventricle abnormalities. However, the clinical significance needs to be confirmed in larger studies.

Reprint requests and correspondence: Dr. Fiorenzo Gaita, Divisione di Cardiologia, Ospedale Civile, Via Botallo, 4-14100 Asti, Italy. E-mail: gaitaf@tin.it.

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